Occupational Lung Disease

Definition

- Occupational lung disorder has be defined as a disease arising out of or in course of employment.

Determinants of inhalational exposure

- Particles size of air contaminates

Particles > 10 -15 um diameter do not penetrate beyond the nose and throat.

- Particles are divided into three size fractions on the basis of their size character and source.

1.Coarse-mode fraction –particles size of 2.5- 10 um contain crustal elements such as silica, aluminum, and iron. Mostly deposit relatively high in the tracheobronchial tree.

2.Fine mode fraction –practical size <2.5 um and carried to the lower airways and get deposited .fine particles are created by burning of fossil fuels or high temperature industrial process, gases ,fumes.

3.Ultra fine fraction - <0.1 um in size deposit in the lung and they come in contact with the alveolar walls ,however particles of this size range may penetrate into the circulation and be carried to extra pulmonary sites.

Classification

•Inorganic (mineral) dust/Pneumoconiosis

silica, asbestos, coal ‹talc, silicates Fe,

barium, tin

•Organic –grain dusts ,cotton , pollens etc

- Immunologic

Allergic alveolitis (hypersensitivity pneumonitis(

Asthma - feathers, enzymes, cotton, platinum

Pneumoconiosis

Definition

may be defined as a permanent alteration of lung structure due to the inhalation of mineral dust and the tissue reactions of the lung to its presence, excluding bronchitis and emphysema.

The most common pneumoconiosis are Coal-workers' pneumoconiosis, Silicosis and Asbestosis but many other types have been described, usually due to inhalation of mixed dusts containing silicates.

Pneumoconiosis

- For clinical pneumoconiosis to develop, 3 essential factors are required:
  - Exposure to specific substance: coal, appear relatively inert and may accumulate in considerable amounts with minimal tissue response; while silica and asbestos, have potent biologic effects.
  - Particles of appropriate size to be retained in lung (1-5µm)
  - Exposure for a sufficient length of time (usually around 10 years)

CLASSIFIED

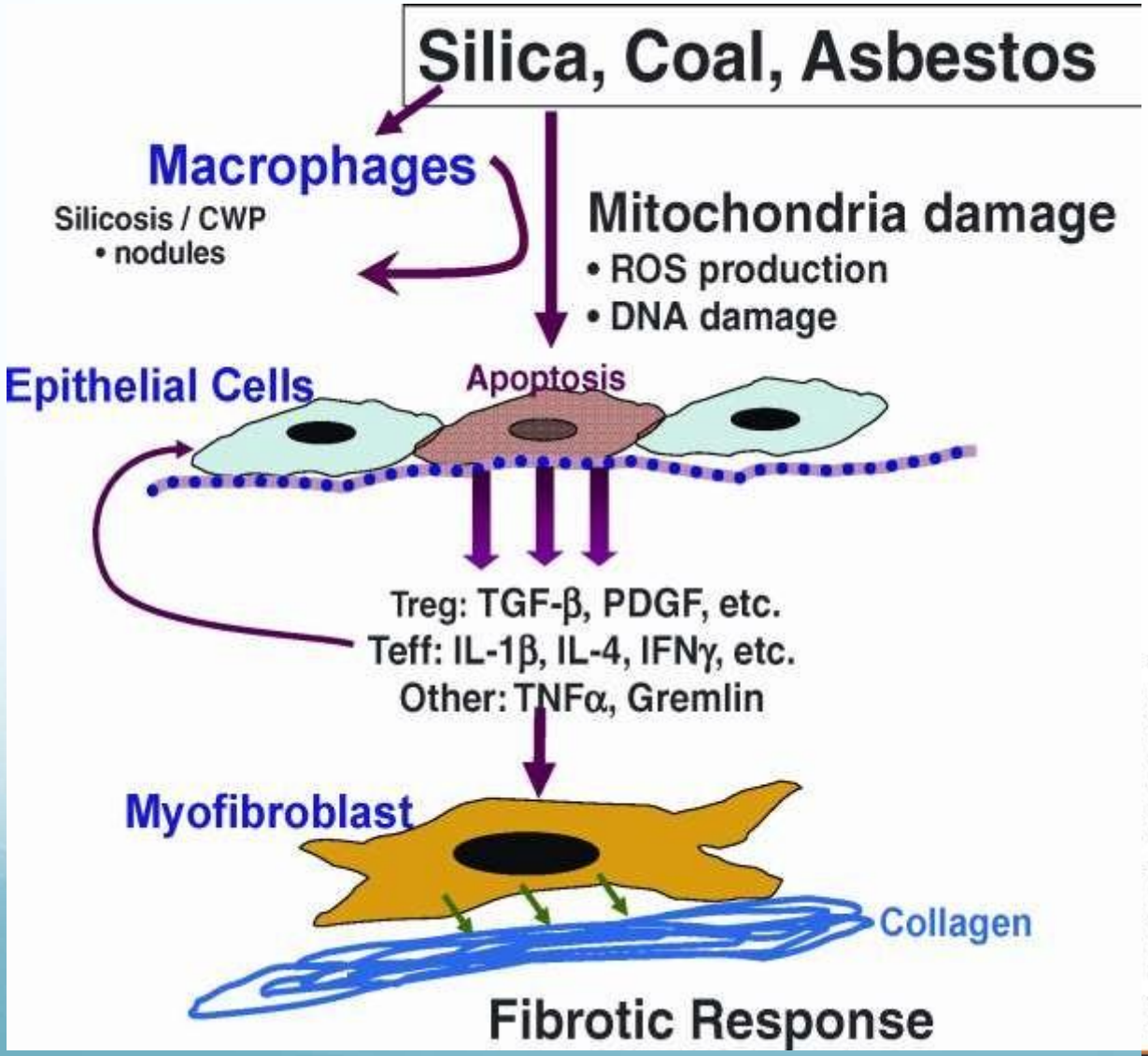
fibrotic(**focal nodular ,diffuse fibrosi)**

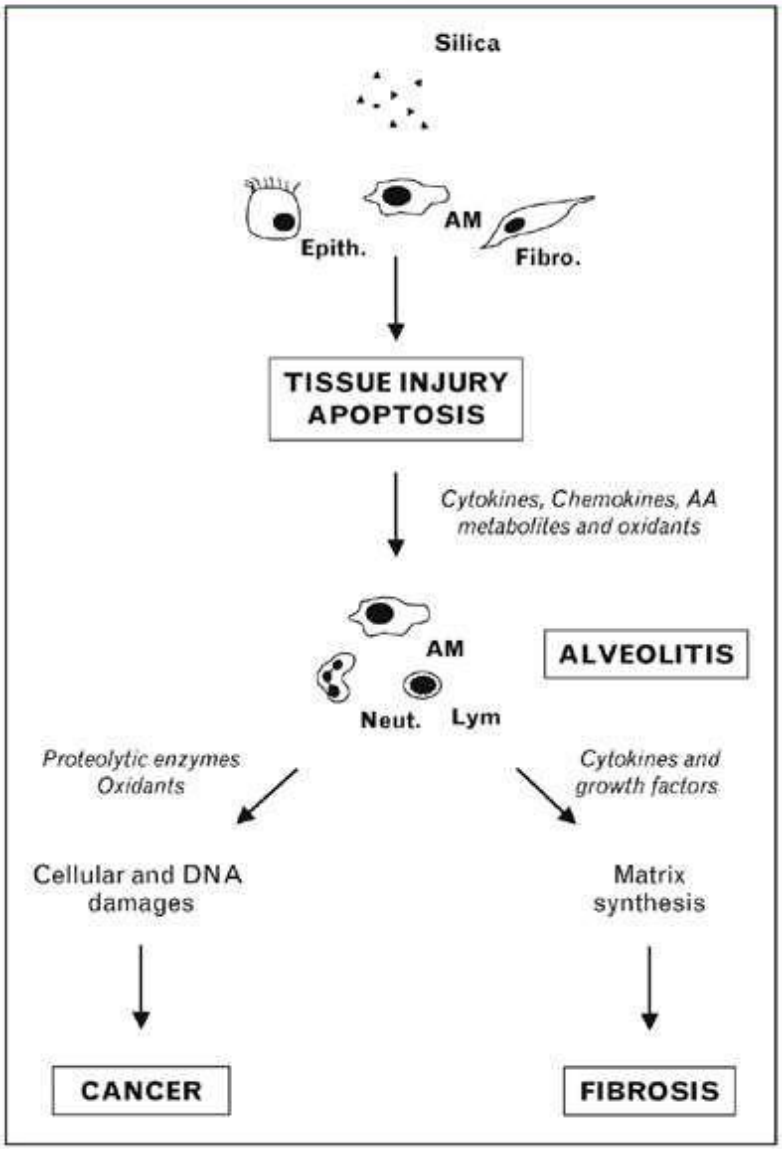
.1silicosis -Nodular fibrosis
2.coal worker pneumoconiosis
3.asbestosis --Diffuse fibrosis
4.berylliosis- Granulomatous reaction

nonfibrotic
(particle-laden macrophages, no fibrosis(

.1siderosis
2.stannosis
3.baritosis

Pathophysiology of pneumoconiosis





Silica particles (▲), that reach the alveolar epithelial surface, induce lung injury and apoptosis and stimulate resident alveolar macrophages (AM), epithelial cells (Epith.) and fibroblasts (Fibro.) to release several factors (chemokines, cytokines, arachidonic acid (AA) metabolites and oxidants) attracting and stimulating leukocytes such as neutrophils (Neut.) and lymphocytes (Lym.) into the lung. The activated alveolar macrophages and neutrophils produce a burden of oxidants and proteases that additionally induce apoptosis and injure the alveolar wall. Oxidants can also induce DNA damage leading to cancer. Leukocytes also release growth factors and cytokines that stimulate lung fibroblasts to proliferate and to produce matrix proteins. The result is more mesenchymal cells and a larger mass of connective tissue matrix, characterising lung fibrosis.

COAL WORKER PNEUMOCONIOSIS

- Pathologic entity resulting from deposition of coal dust in the lungs.

- **Simple cwp** – with prolonged exposure to coal dust for 15 to 20 years ,small rounded opacities develop usually not associated with pulmonary impairment.
- **Complicated cwp** – appearance of nodules> 1cm in diameter on chest radiography usually confined to upper half of the lungs . And it progresses to PMF(Progressive massive fibrosis) that is accompanied by severe lung deficits causing chronic bronchitis and COPD.
- When exposure is terminated the simple type will not progress; PMF type will progress

CAPLANS SYNDROME

- **coal workers with rheumatoid disease may develop nodules even after relatively low exposures to dust.**
- **The lesions are typically subpleural.**
- ***The lesions may grow rapidly, appear in crops(in contrast to silicotic/CWP nodules that appears over a period of time) cavitate and produce a pneumothorax***

Clinical Features of CWP

- Chronic cough and sputum production are more common with increasing dust-exposure, regardless of the presence or absence of simple pneumoconiosis.
- Some miners with simple pneumoconiosis may have no related symptoms or physical signs, but with severe airflow obstruction or advanced pneumoconiosis, dyspnea, cough, and sputum production are frequent. Edema of the lower extremities, and findings consistent with corpulmonale may occur.
- Melanoptysis (expectoration of black sputum) occasionally results from excavation of a PMF lesion

- Atypical cases included crackles, finger clubbing, restrictive impairment, diffusion block, and neutrophilic bronchoalveolar lavage (BAL)
- CWP has not been associated with increased risk for development of coexisting mycobacterial infection,

Simple CWP

- With prolonged exposure to coal dust for 15 to 20 years, small rounded opacities develop usually not associated with pulmonary impairment
- Asymptomatic & is often a radiographic diagnosis
- When exposure is terminated the simple type will not progress

Complicated CWP

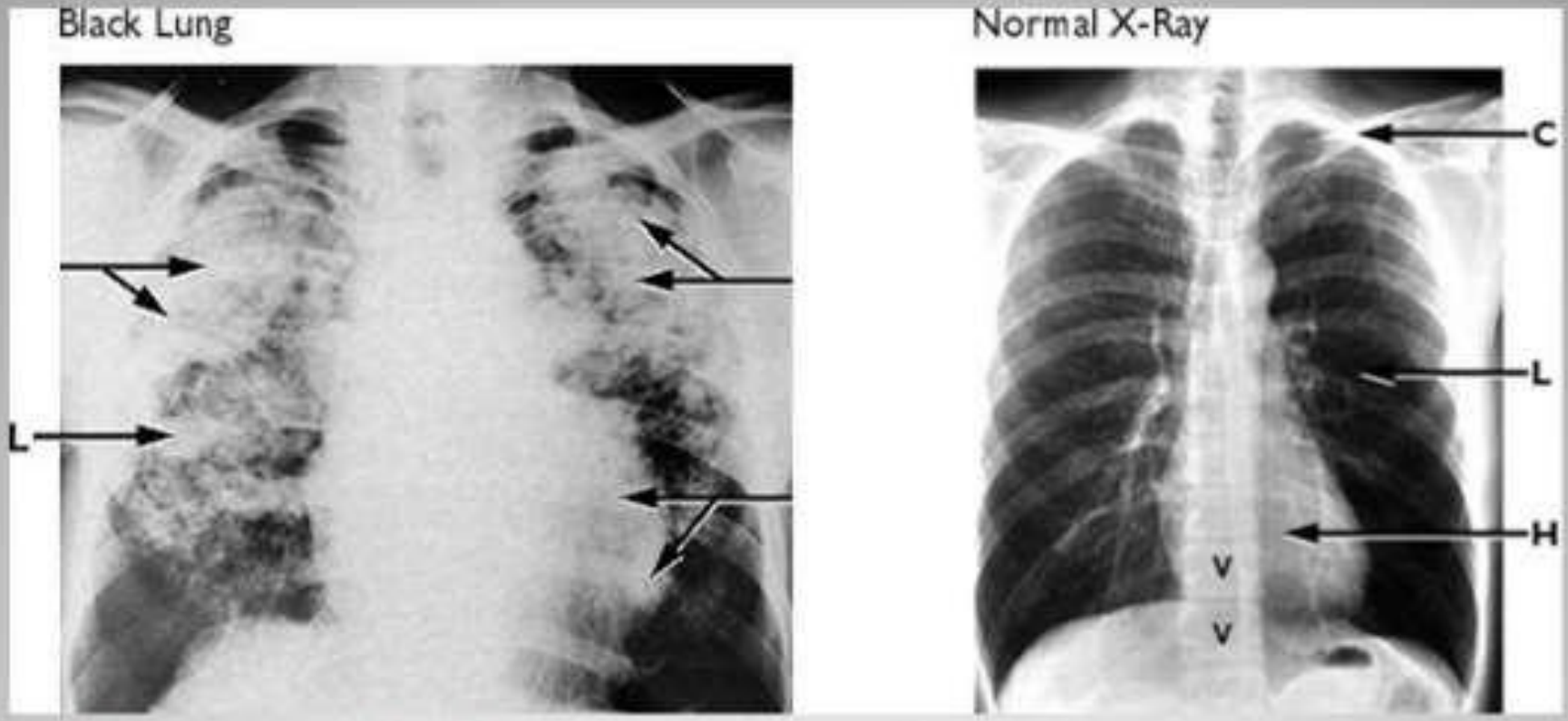
- Appearance of nodules > 1cm in diameter on chest radiography usually confined to upper half of the lungs.
- progresses to PMF, that is accompanied by severe lung deficits chronic bronchitis and COPD

Chest radiography

- Earliest sign of CWP is nodular shadowing on the chest radiograph.
- These appearances are classified according to ILO standard
- They are usually more profuse in the upper and middle zones
- The early nodular lesions are frequently accompanied by kerley B lines.
- This condition presents as a larger shadow, equal to or greater than 1cm in maximum diameter, often initially in the right upper zone. The lesion gradually grows, becomes more radiodense and causes distortion of adjacent lung and bronchi often with bullous emphysema.
- Ultimately it is completely replaced by PMF and emphysema, with resultant cor pulmonale
- HRCT may reveal parenchymal nodules and emphysema when standard radiographs are normal. In atypical cases, CT scans may show ground-glass
- Opacities and honeycombing, at times without nodular findings typical of CWP.

Caplan’s syndrome

- A few miners, especially those with rheumatoid arthritis or with rheumatoid factor in their blood, develop well defined rounded lesions that grow to about 2–3cm in diameter or rarely somewhat larger
- They are usually multiple and have a marked tendency to cavitate.
- They often occur on a background of no simple pneumoconiosis and in miners with a relatively low dust exposure.
- They are called Caplan’s lesions and their combination with rheumatoid disease
- They do not have an ominous prognosis and cause no significant functional impairment, being unassociated with emphysema



An abnormal chest x-ray shows severe scarring (arrows) in the lungs (L) caused by progressive massive fibrosis. This finding is seen in severe black lung disease caused by exposure to coal dust

Diagnosis & Management

- A history of occupational exposure to coal and a chest radiograph are the fundamental elements in the diagnosis of CWP.(CT when chest X-ray insensitive)
- Treatment of latent tuberculosis infection should be considered for coal workers who are thought to have had significant silica dust exposure or who have evidence of silicosis

- Smoking-cessation counseling
- Management of chronic bronchitis and emphysema

SILICOSIS

- It is a type of pneumoconiosis: condition characterized by accumulation of silica dust in lung parenchyma & tissue reaction to it
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- Also known as **Potter's rot**, is a form of occupational lung disease caused by inhalation of crystalline silica dust & is marked by inflammation & scarring in forms of nodular lesions in upper lobes of the lungs.
- Silicon dioxide or silica is the most abundant compound in the earth's crust, where it is mostly found in the crystalline form known as quartz.

SILICOSIS

Occupations in which exposure to quartz take place are:

- Mining
- Tunnelling
- Quarrying
- Stonemasonry
- Sandblasting
- Fettling and foundry work
- Ceramics
- Brick-making
- Refractory kiln repair
- Silica flour manufacture
- Abrasive manufacture
- Diatomaceous earth manufacture
- ,Glass and cement manufacturing

SILICOSIS

Main determinants of silicosis

- Silica dust
- Its duration exposure
- Size of particle
- Distribution & respirability of airborne particles
- Their fibrogenic potential

Classification of Silicosis

- **Acute-** which develops over months in response to exceedingly heavy exposures as in sandblasting or dry drilling. It is characterized by appearances suggestive of pulmonary oedema. Acute enlargement of the hilar nodes may occur with heavy exposure to quartz in individuals without prior exposure

- **Accelerated-** associated with heavy exposures over a relatively short period of a few years (typically 5–10 years), (, presents radiologically as progressive irregular upper zone fibrosis, sometimes with relatively sparse and indistinct nodularity

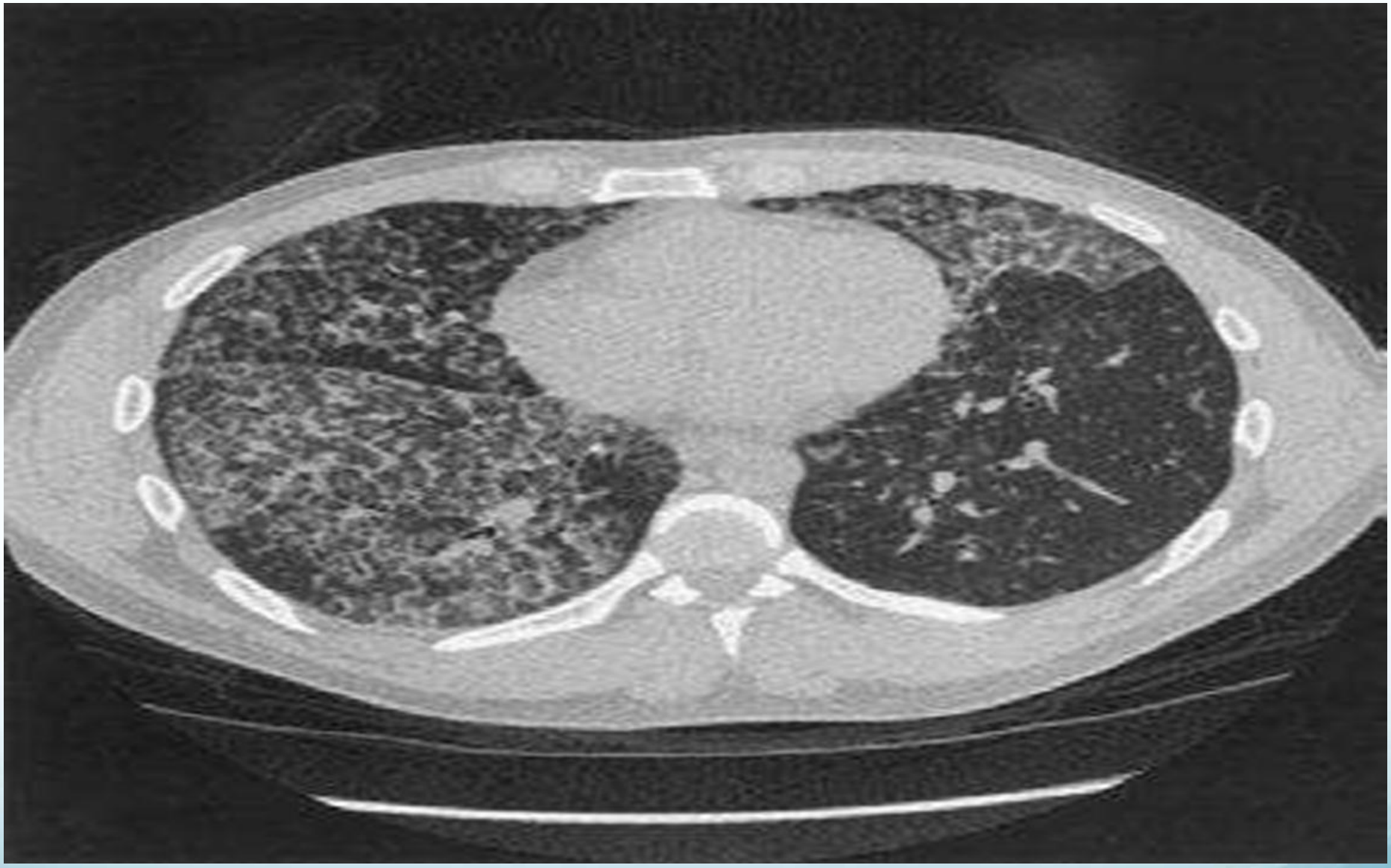
- **Chronic silicosis**

Commonest form(10–20 years)

- Occurs after many decades of exposure to relatively low levels of silica.
- Characterized by gradually progressive dyspnea, dry cough & evidence of progressive fibrosis of both lungs on chest X-Ray

Acute silicosis

- The clinical and pathologic features of acute silicosis are similar to those of pulmonary alveolar proteinosis
- The chest radiograph may show profuse miliary infiltration or consolidation, and there is a characteristic HRCT pattern known as “crazy paving”
- The disease may be quite severe and progressive despite the discontinuation of exposure. Whole-lung lavage may provide symptomatic relief and slow the progression.

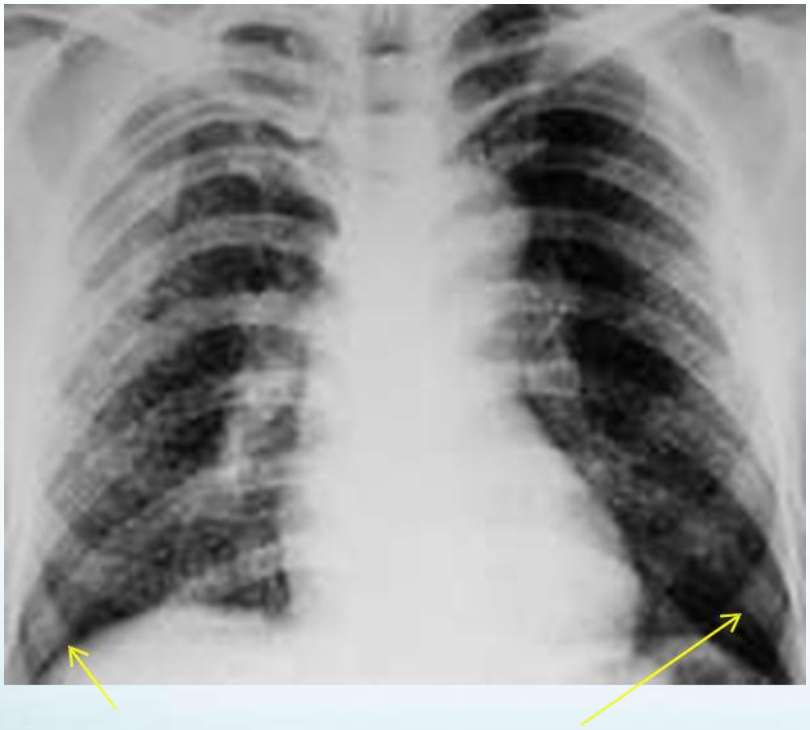


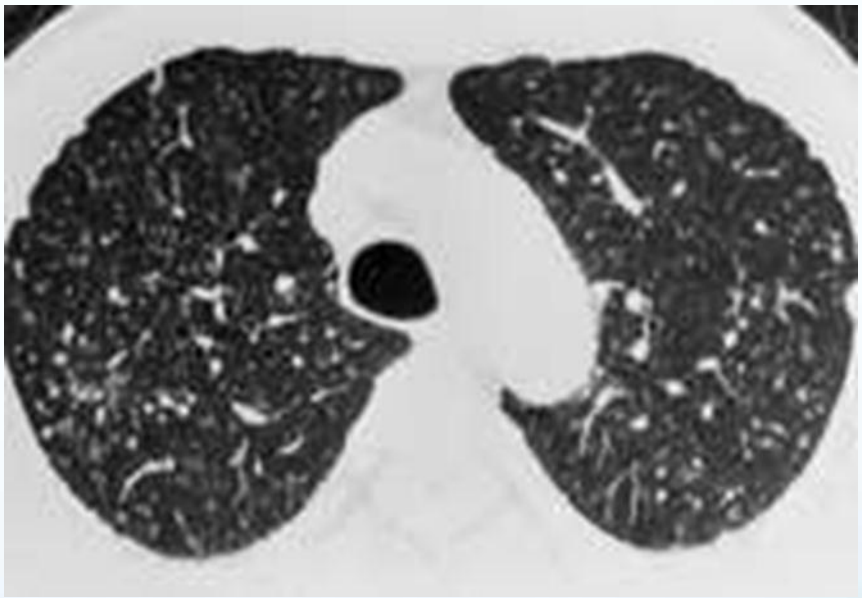
Chronic silicosis

- Chronic simple : With long-term, less intense exposure, small rounded opacities in the upper lobes may appear on the chest radiograph after 15–20 years of exposure, usually without associated impairment of lung function (simple silicosis)

- Calcification of hilar nodes may occur in as many as 20% of cases and produces a characteristic “eggshell” pattern. Silicotic nodules may be identified more readily by HRCT

Simple silicosis





Complicated silicosis

- The nodular fibrosis may be progressive in the absence of further exposure, with coalescence and formation of nonsegmental conglomerates of irregular masses >1 cm in diameter (*complicated silicosis)*

- These masses can become quite large, and when this occurs, the term *progressive massive fibrosis (PMF) is applied*

- *Significant functional* impairment with both restrictive and obstructive components may be associated with PMF.

DIAGNOSIS

There are 3 key elements to diagnosis of silicosis

- Patient history should reveal exposure to sufficient silica dust to cause this illness.
- Chest X-Ray revealing findings consistent with silicosis.
- There are no underlying illnesses that are more likely to be causing the abnormalities

- Physical examination is usually unremarkable unless there is complication. PFT may reveal airflow limitation, restrictive defects, ↓ diffusion capacity, mixed defects or may be normal

- For uncomplicated silicosis, chest x-ray will confirm presence of small (<10 mm) nodules in lungs, especially in upper lung zones

Complications

SILICOTUBERCULOSIS

- Patient with silicosis are 3 fold increase risk for TB infection
- Both humoral & CMI response are inhibited in silicosis.
- mycobacteria use iron stored by the silica particles

- Pulmonary complications of silicosis also include chronic bronchitis & airflow limitation, fungal lung infections, compensatory emphysema & pneumothorax.
- There are some data revealing association between silicosis & autoimmune diseases (Nephritis, Scleroderma & SLE) especially in acute or accelerated silicosis.
- Esophageal compression
- Atelectasis
- PAH,Chronic respiratory failure, Chronic cor pulmonale
- Recurrent chest infection
- Lung abscess ,Hydro pneumothorax
- Lung cancer

Management

- Silicosis is an irreversible & progresive condition with no cure.
- Treatment options currently focus on alleviating symptoms & preventing complications including stopping further exposure to silica & other lung irritants, including tobacco smoking, cough suppressants, antibiotics for bacterial lung infection, ATT for those with active TB, chest physiotherapy to help bronchial drainage of mucus, O_2 administration to treat hypoxemia, bronchodilators to facilitate breathing.
- Lung transplantation to replace damaged lung tissue is the most effective treatment

ASBESTOS-RELATED DISEASES

ASBESTOS-RELATED DISEASES

- Asbestos is a generic term for several different mineral silicates, including chrysolite (most widley used), amosite, anthophyllite, and crocidolite.
- Occupation in the production of asbestos products (mining, milling, and manufacturing), shipbuilding and construction trades including pipe fitters
- People are exposed to asbestos anywhere in the chain from the mine and crusher, the site of production of materials to the place where the materials are used. Indeed, exposure to waste asbestos material at dumps is another area of concern
- The major health effects from exposure to asbestos are pleural and pulmonary fibrosis, cancers of the respiratory tract and pleural and peritoneal mesothelioma

Asbestos-related diseases

Benign

Pleural diseases

.1Plaques

.2Diffuse pleural thickening

3.Effusion

.4Calcification

Parenchymal diseases

1.Asbestosis [parenchymal fibrosis caused by asbestos inhalation[

2.Rounded atelectasis

3.Benign fibrotic masses

4.Transpulmonary bands

Malignancy

1.Malignant mesothelioma

2.Bronchogenic carcinoma

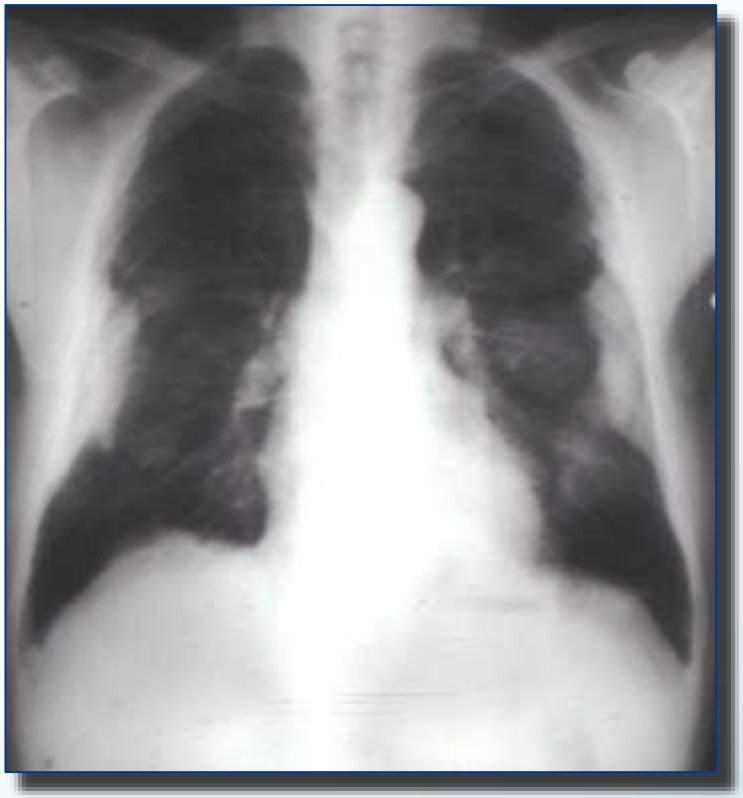
Asbestos-Related Pleural Abnormalities

- Four types of abnormalities:
  - Pleural plaques the most common manifestation of past asbestos exposure
  - Benign asbestos pleural effusions
  - Diffuse pleural thickening
  - Pleural disease puts patient at risk for other asbestos related diseases – 10% get interstitial fibrosis within 10 years

- Mostly asymptomatic, though some can cause dyspnea or cough
- Latency periods: 10-30 years(shorter latency is for pleural effusion)
- No specific threpaies
- Pleurectomy in severe cases.

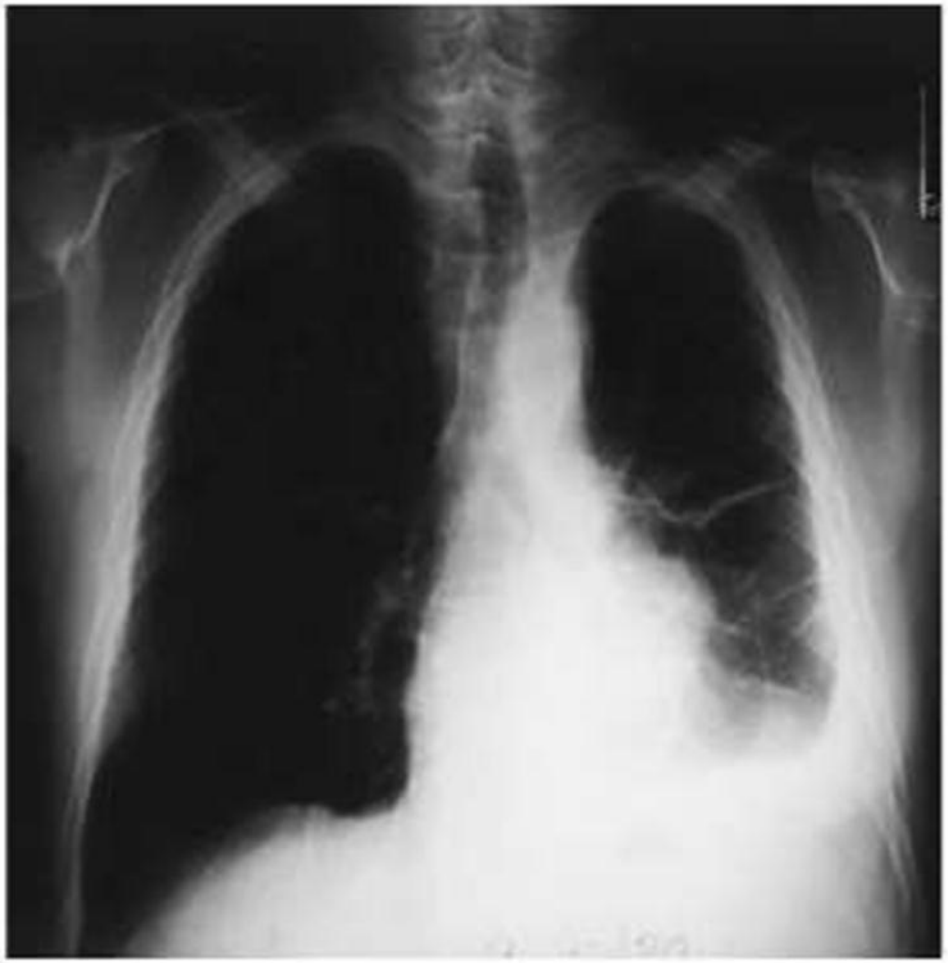
Chest Radiograph Findings: Asbestos-Related Pleural Abnormalities

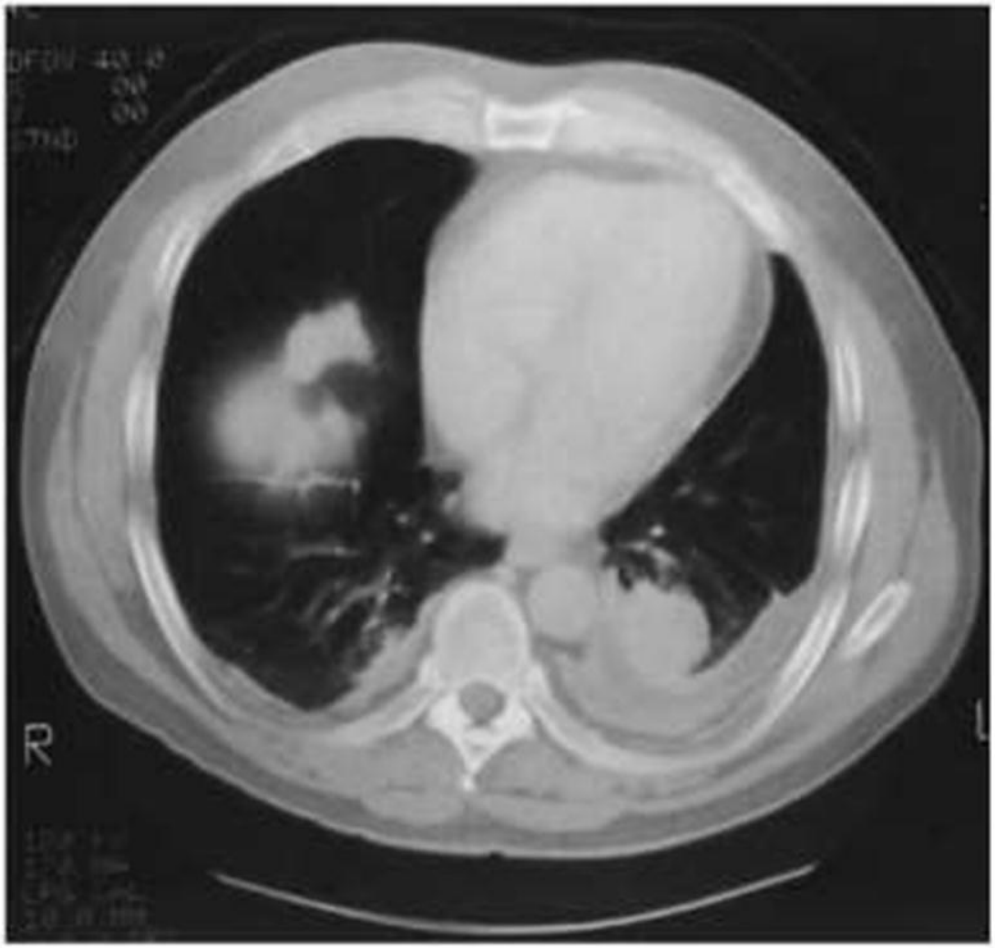
- Pleural plaques
  - Areas of pleural thickening
  - Sometimes with calcification
- Pleural effusions
- Diffuse pleural thickening
  - Lobulated prominence of pleura adjacent to thoracic margin
  - (over ¼ of chest wall(
  - Interlobar tissue thickening
- Rounded atelectasis
  - Rounded pleural mass
  - Bands of lung tissue radiating outwards



Rounded Atelectasis

- Caused by scarring of the visceral and parietal pleura and the adjacent lung, with the pleural reaction folding over on itself.
- The pleural surfaces then fuse to one another, trapping the underlying lung and leading to atelectasis.
- As a result of this alteration, a mass lesion that mimics lung cancer can be seen on the PA chest radiograph
- This lesion is most easily appreciated to be a pseudotumor with use of CT scanning.
- HRCT can noninvasively demonstrate continuity to areas of diffuse pleural thickening, evidence of volume loss in the adjacent lung, or a characteristic comet tail of vessels and bronchi sweeping into wedge-shaped mass .
- HRCT scans localized most cases of rounded atelectasis to the lower, posterior portion of the lung (right).





The chest radiograph *shows* left-sided pleural effusion, bilateral pleural thickening, greater on the left than on the right, and a mass in the left midlung field.

HRCT *demonstrates the mass to be rounded atelectasis, with* bronchovascular structures entering the trapped lung. It also reveals the pleural effusion, bilateral pleural thickening.

Parenchymal Asbestosis

- Diffuse interstitial fibrosis with:
  - Associated more with crocidolite
  - Smokers more prone to disease and XRC interstitial infiltrates
- Radiographic changes: >10 years
- Latency period: 20-40 years

Lung Carcinoma

- Latency period: 20-30 years
- Bronchogenic Ca: 5x higher incidence in non-smoking asbestos workers, 90x higher in smoking asbestos workers.
- Adeno ca is most common.
- Chrysotile highest risk bronchogenic Ca.

Malignant Pleural Mesothelioma

- Tumor arises from the thin pleural membrane surrounding the lungs
- Rapidly invasive
- Rare, although incidences are increasing
- Long latency period: Usually 30-40 years

BERYLLIUM

- Beryllium – is a light weight metal
- **Exposure** –manufacture of alloys, ceramics, or high technology electronics , nuclear reactors

- Beryllium may produce
  -Acute pneumonitis‘
  -Chronic granulomatous inflammatory disesase that is similar to sarcodisis.
  -Lung cancer
- **Pathogenesis** is a result of delayed type hypersensitivity reaction stimulating proliferation of T-cells leading to inflammatory ,fibrosis and granuloma formation.
-

- Clinical features- cough , chest pain, arthralgia's ,fatigue and weight loss

- **BeLPT[beryllium lymphocyte proliferation test]-**blood is drawn and in the lab, the WBC are separated from the rest of the blood cells and then mixed with beryllium solution. If the immune system is sensitized to beryllium, the cells will multiply, producing an abnormal BeLPT result.In normal individuals cells will not multiply.

- Fiberoptic bronchoscopy with transbronchial lung biopsy is required to make diagnosis of CBD . Biopsy shows noncaseating granulomas or monocytic infiltration in lung tissue.

Other occupation lung disease

Disease	Source of Exposure	Major Antigen
Farmer's lung	Moldy hay	*Saccharopolyspora rectivirgula*
Bagassosis	Moldy sugar cane fiber	*Thermoactinomyces sacchari*
Grain handler's lung	Moldy grain	*S rectivirgula, T vulgaris*
Humidifier/air-conditioner lung	Contaminated forced-air systems, heated water reservoirs	*S rectivirgula, T vulgaris*
Bird breeder's lung	Pigeons, parakeets, fowl, rodents	Avian or animal proteins
Cheese worker's lung	Cheese mold	*Penicillium casei*
Malt worker's lung	Moldy malt	*Aspergillus clavatus*
Paprika splitter's lung	Paprika dust	*Mucor stolonifer*
Wheat weevil	Infested wheat	*Sitophilus granarius*
Mollusk shell hypersensitivity	Shell dust	Sea snail shells

FARMER'S LUNG

- Caused by inhalation of spores of thermophilic actinomycetes, higher bacteria of a filamentous appearance that grow in hay or other organic matter that has been stored in a damp condition

- Spores of Thermophilic actinomycetes produce a hypersensitivity pneumonitis

MUSHROOM WORKER'S LUNG

- Mushrooms are grown commercially on compost, often made of straw and horse droppings that is allowed to ferment, which is then heated in moist air to just below 60°C

- While these conditions suit the mushroom mycelia with which it is seeded, they are also ideal for the growth of thermophilic actinomycetes

HYPERSENSITIVITY PNEUMONITIS

- Also referred to as extrinsic allergic alveolitis
- Pulmonary disease that occurs due to inhalational exposure to a variety of antigens leading to an inflammatory response of the alveoli and small airways.
- Systemic manifestations such as fever and fatigue can accompany respiratory symptoms.
- Sensitization to an inhaled antigen as manifested by specific circulating IgG antibodies is necessary for development of HP
- Causes of HP include farmer's lung, bagassosis, mushroom workers lung, Air conditioner and humidifier lung, woodworkers lung

HYPERSENSITIVITY PNEUMONITIS

The presentation of HP varies from an acute form to a more indolent pattern in accordance with the antigen load
High antigen load : influenza-like symptoms accompanied by cough, breathlessness and wheeze
low-level antigen: will typically present in a more indolent fashion with slowly progressive breathlessness; in some cases, established fibrosis may be present by the time the disease is recognised.
Chest auscultation typically reveals widespread end-inspiratory crackles and squeaks.

HYPERSENSITIVITY PNEUMONITIS

Investigations

In cases of acute HP, the chest X-ray typically shows ill-defined patchy airspace shadowing, which, given the systemic features, may be confused with pneumonia. HRCT is more likely to show bilateral ground-glass shadowing and areas of consolidation superimposed on small centrilobar nodular opacities with an upper and middle lobe predominance (Fig. 17.63). In more chronic
disease, features of fibrosis, such as volume loss, linear opacities
and architectural distortion, appear. In common with other fibrotic
diseases, pulmonary function tests show a restrictive ventilatory defect with reduced lung volumes and impaired gas transfer, dynamic tests may detect oxygen desaturation and, in more advanced disease, type I respiratory failure is present at rest.

Diagnosis

17.82 Predictive factors in the identification of hypersensitivity pneumonitis

- Exposure to a known offending antigen
- Positive precipitating antibodies to offending antigen
- Recurrent episodes of symptoms
- Inspiratory crackles on examination
- Symptoms occurring 4–8 hours after exposure
- Weight loss

Treatment

- Avoidance
Cessation of exposure
Dust mask
Steriod in acute face

Occupational asthma

Occupational asthma should be considered in any individual of working age who develops new-onset asthma, particularly if the individual reports improvement in asthma symptoms during periods away from work, e.g. at weekends and on holidays. Workers in certain occupations appear to be at particularly high risk (Box 17.79) and the condition is more common in smokers and atopic individuals. Depending on the intensity of exposure, asthmatic symptoms usually develop within the first few years of employment but are classically preceded by a latent period. Symptoms of rhinoconjunctivitis often precede the development of asthma. When occupational asthma follows exposure to high-molecular-weight proteins, sensitisation may be demonstrated by skin testing or measurement of specific IgE to the agent in question

Occupational asthma

. Confirmation of occupational asthma
should be sought from lung function tests. This usually involves serial recording of peak flow at work at least four times per day for a minimum of 3 weeks and, if possible, including a period away from work . In certain circumstances, specific challenge tests are required to confirm the diagnosis
It may be possible to remove the worker from the implicated agent, but when this cannot be done, consideration of personal protective equipment and workplace hygiene may allow the worker to retain their job and livelihood. Specialist follow-up in such situations is highly advisable. A favourable prognosis is indicated by a short history of symptoms and normal lung function at diagnosis. Where reduction or avoidance of exposure fails to bring about resolution
the management is identical to that of any patient with asthma

THANK YOU